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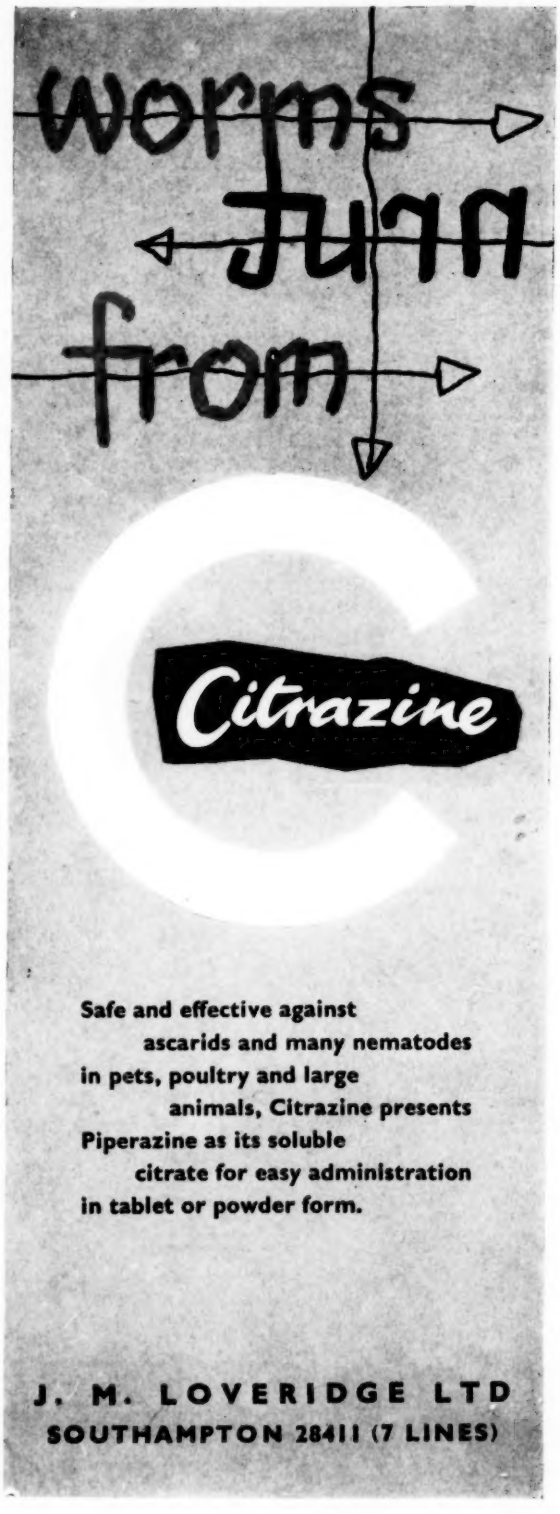

THE VETERINARY RECORD

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Epiphysial Fusion in the Greyhound

BY

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AND

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SUMMARY.—An analysis is presented of the changes shown by radiology which are undergone by each joint in the fore- and hind-limbs of developing greyhounds. It is pointed out that complete fusion of the epiphyses is the easiest stage to recognise, but this is not necessarily coincident with cessation of longitudinal growth.

Introduction

THE use of radiography to study the fusion of the epiphyses of the dog is a fairly recent innovation, although it has been used extensively over a long period for man. In 1948 Seoudi presented his results on the Egyptian Armant breed and in 1954 and 1957 Pomriaskinsky-Kobozieff *et al.* published their work on the lower parts of limbs. While this present article was being prepared, Hare (1959) has published his results on the fore limb. Seoudi's work is based on 8 dogs : although he does not mention the intervals at which they were examined the times he quotes are given in months. Pomriaskinsky-Kobozieff *et al.* based their work on a Cocker Spaniel, a litter of 7 Alsatians and 2 mongrels X-rayed at stated intervals. In addition, " le service de radiologie comparée de l'École d'Alfort nous a donné la possibilité d'étudier un grand nombre de radiographies de tout âge et de toutes races." However, these authors were mainly interested in the appearance of carpal and tarsal centres although times of fusion of the lower bones were also recorded. Hare (1959) has used 8 dogs for epiphysial union from 2 breeds (1 of which was a Scotch Collie, the other was not specified) and although it is stated that radiographs were made at regular intervals he does not give more specific details. The data he presents are based on his own observations and those of previous workers, making it impossible to deduce his own findings, and resulting in periods of 2 to 3 months being given for the times of fusion.

In addition to these works, Schlotthauer and Janes (1952) give some data on the closure of epiphyses at the femoro-tibial joint and in the same year correspondence in THE VETERINARY RECORD drew attention to radiological appearances of the developing stifle joint (Turnbull, Bateman, 1952).

Materials and Methods

Twenty-eight greyhounds from 6 litters were used in this survey. The distribution of sexes is given in Table I.

TABLE I

THE NUMBER OF DOGS AND THE DISTRIBUTION OF SEXES IN THE SIX LITTERS USED IN THE SURVEY

Litter	*Males*	*Females*
H.	1	3
S.	2	1
K W.	4	3
W.M.	3	3
W.P.	4	1
E.	3	0

The earliest examinations were made at 13 weeks and thereafter every fortnight : towards the end of the series this interval was extended to 4 weeks. It was not possible to adhere rigidly to this time-table and so all times quoted should be understood to mean ± 1 week. (See Table II.)

The radiographs of each region were compared and an example typical of each age was selected and a tracing made. This article is a report on the typical results. A study of the variation within each litter and between litters, will be presented later.

TABLE II
THE NUMBER OF DOGS USED AT EACH EXAMINATION AND THEIR AGES

Week of examination ± 1 week	Number of dogs	Week of examination ± 1 week	Number of dogs
13	4	35	7
15	4	37	4
17	10	39	6
19	20	41	7
21	14	43	4
23	11	45	8
25	11	47	7
27	14	51	5
29	11	55	4
31	11	59	6
33	21		

Observations

Shoulder Joint (Fig. 1)

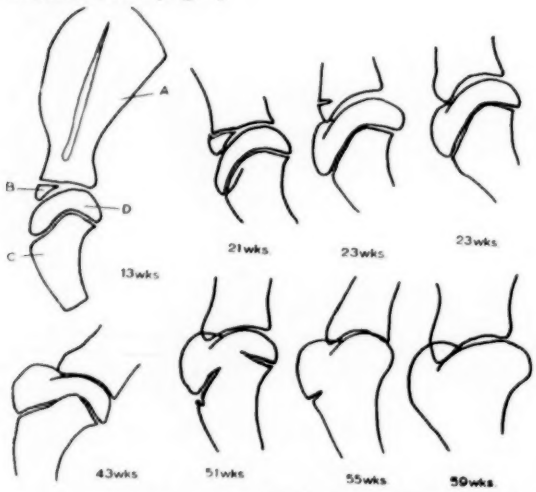

FIG. 1.—Lateral views of the developing shoulder joint. A : scapula. B : epiphysis for tuber scapulae. C : humerus, diaphysis. D : humerus, proximal epiphysis.

The scapula has an epiphysis which forms the tuber scapulae for the origin of the biceps muscle. At 13 weeks this epiphysis is attached only by cartilage to the remainder of the scapula. At 23 weeks some dogs show some of this cartilage juncture still present, other dogs show that the epiphyseal union is complete. No cartilage is present at 25 weeks.

At 13 weeks the proximal end of the humerus has an epiphysis sitting as a cap on the end of the diaphysis. The cartilage plate is complete up to about 43 weeks when some bony union is present near the centre. At 51 weeks remnants of cartilage can only be seen at the periphery and 4 weeks later only at the dorsal edge of the shaft. This area of cartilage has disappeared by 59 weeks.

Elbow Joint (Fig. 2)

At 13 weeks the humerus has a distal epiphysis for longitudinal growth and an epiphysis forming the

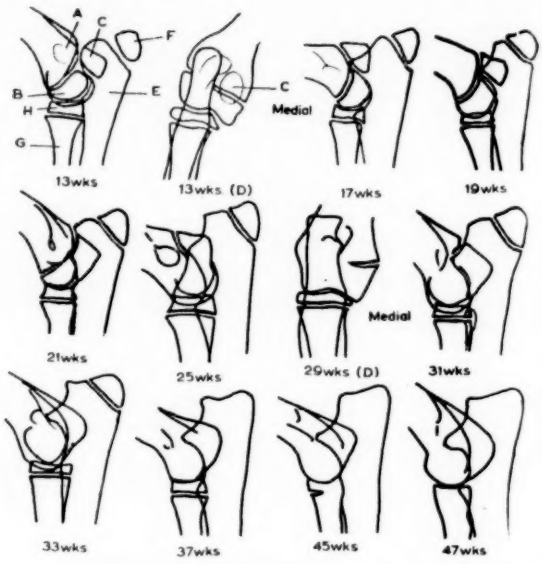

FIG. 2.—Developing elbow joint. (D) Dorsal views, remainder lateral. A : humerus, diaphysis. B : humerus, distal epiphysis. C : humerus, medial epicondyle. E : ulna, diaphysis. F : ulna, proximal epiphysis. G : radius, diaphysis. H : radius, proximal epiphysis.

medial epicondyle for the attachment of the flexor muscles of the lower part of the limb. The ulna has an epiphysis to which the triceps mass is attached. The proximal end of the radius is represented by a single epiphysis.

The 2 epiphyses seen at the distal end of the humerus at 13 weeks remain separate up to the 21st week when they appear to have fused with each other. It also seems that the distal epiphysis has partly fused to the diaphysis at the 21st week and by 25 weeks this is definitely so. The only remnant of cartilage is between the medial epicondyle and the diaphysis. Although present at 31 weeks this has gone by the 33rd week.

The epiphysis of the ulna appears as a cap separated by cartilage from the remainder of the ulna in all the dogs up to the 37th week, when the cartilage plate seems to have vanished. The proximal epiphysis of the radius remains separate for rather longer : at 45 weeks part of the epiphysis has fused to the diaphysis and by 47 weeks no separation can be seen.

Carpal Joint (Fig. 3)

The epiphyses in this joint belong to the radius, ulna and accessory carpal bone.

The epiphysis for the accessory carpal bone is the first to fuse and although separate at 21 weeks has fused by 23 weeks. The distal epiphyses of the radius and ulna remain separate from their diaphyses up to the 45th week. At 47 weeks no separation can be seen.

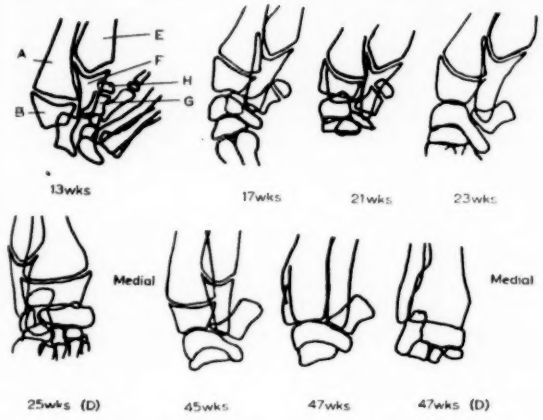

FIG. 3.—Developing carpal joint. (D) Dorsal views, remainder lateral. A : radius, diaphysis. B : radius, epiphysis. E : ulna, diaphysis. F : ulna, epiphysis. G : accessory carpal, body. H : accessory carpal, epiphysis.

The Joints of the Fore Paw (Fig. 4)

The metacarpal bones only have distal epiphyses and the first and second phalanges only proximal epiphyses. The third phalanx has no epiphysis.

In the region of the paw we find the phalangeal epiphyses unite by the 27th week whereas the epiphysis of the metacarpus remains unfused until the 31st week when no remnant of the cartilage plate can be seen. These observations apply to the digits 2 to 5 inclusive and at the intervals used it appears that fusion takes place at the same time in all these digits.

Hip Joint (Fig. 5)

Observations were made on the proximal end of the femur only and not on the acetabular region of the pelvis. Three femoral epiphyses are found in this joint : the proximal epiphysis for longitudinal growth, and epiphyses for the greater (lateral) trochanter and lesser (medial) trochanter which serve for muscular attachment.

It is unusual to see the cartilage plates for all these epiphyses on any one radiograph of one joint. The limbs were placed in different positions on the 2 sides of the body while a ventrodorsal picture was taken. In this way most radiographs showed the epiphyses in the one or the other hip joint.

All the epiphyses can be seen separated up to the 15th week. At 17 weeks the greater trochanter appears fused in part to the proximal end of the femoral shaft but a segment of cartilage plate is still present. This condition continues till the 43rd week.

On several radiographs at different ages the proximal epiphysis of the femur appears fused in part to the diaphysis (for example, the illustrations representing 13 weeks). The appearance is usually due to the distortion produced in radiographs of those areas of the body where positioning is difficult. It does not seem possible to decide when actual fusion first occurs : it is possible to decide when only a small part of the epiphysial plate is present at one edge only.

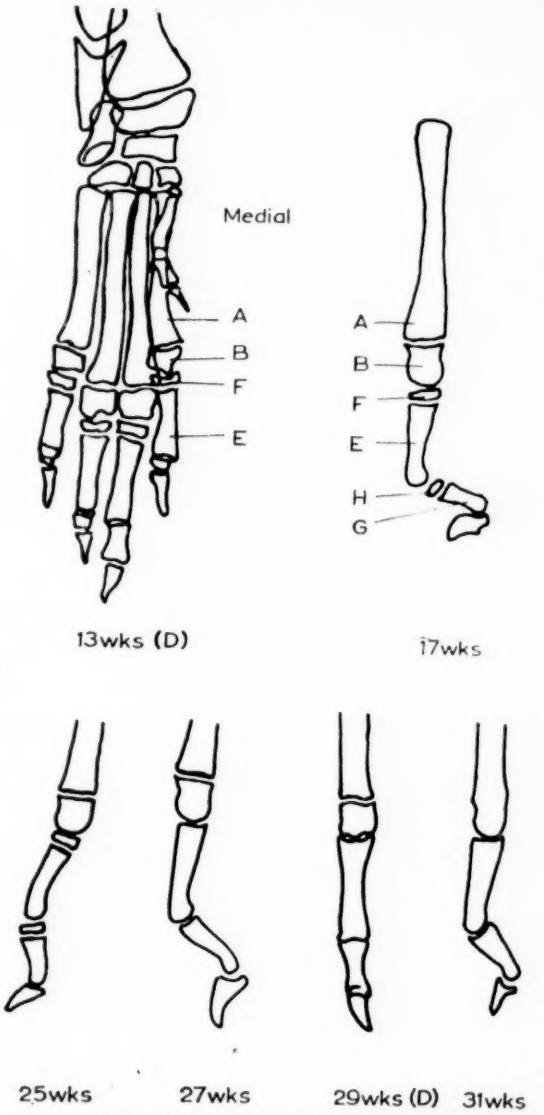

FIG. 4.—Developing fore-paw. (D) Dorsal views, remainder lateral. The first tracing is of the whole paw, the remainder are of one metacarpal and digit only. A : metacarpal, diaphysis. B : metacarpal, epiphysis. E : first phalanx, diaphysis. F : first phalanx, epiphysis. G : second phalanx, diaphysis. H : second phalanx, epiphysis. It is not always possible to see the epiphysial plates of the second phalanges on a dorsal view.

On some radiographs at 45 weeks a very small part of the epiphysial plate separating off the proximal epiphysis can be seen, on others fusion is complete. The lesser trochanter appears to be still separate. On later radiographs, 47 weeks *et seq.*, no cartilage remnants can be seen for any of the epiphyses. The proximal epiphysis and greater trochanteric epiphysis are thus fused at about 45 weeks and the lesser trochanter at about 47 weeks.

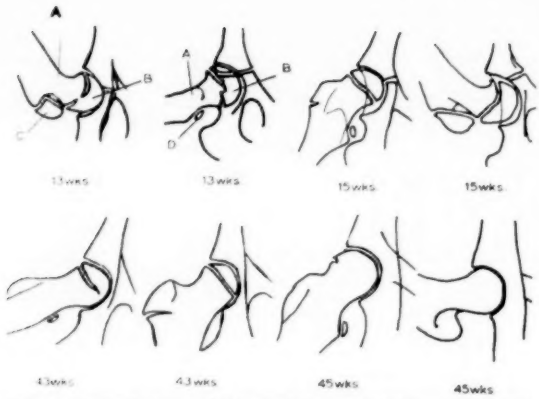

FIG. 5.—Dorsal views of the developing hip joint. A : femur, diaphysis. B : femur, proximal epiphysis. C : femur, epiphysis for greater trochanter. D : femur, epiphysis for lesser trochanter.

Stifle Joint (Fig. 6)

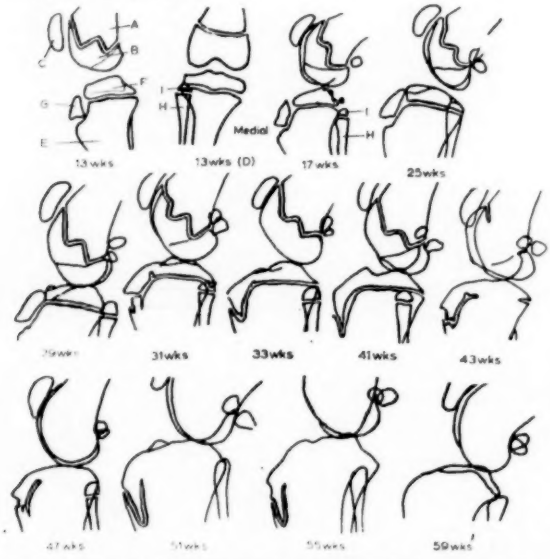

FIG. 6.—Developing stifle joint. (D) Dorsal view, remainder lateral. A : femur, diaphysis. B : femur, distal epiphysis. C : patella. E : tibia, diaphysis. F : tibia, proximal epiphysis. G : tibia, epiphysis for tubercle. H : fibula, diaphysis. I : fibula, proximal epiphysis.

The femur has one epiphysis at the distal end : the tibia has a proximal epiphysis and an epiphysis for the tibial tubercle : the fibula has a proximal epiphysis.

The femoral epiphysis is separated from the diaphysis up to the 41st week but at 43 weeks most of the cartilage plate has disappeared leaving only a small section on the dorsal surface of the bone. This has disappeared by the 47th week and complete fusion has then taken place.

The two epiphyses at the proximal end of the tibia remain separate up to about the 25th week when the tibial tubercle and the proximal epiphysis become fused over a small area. This fusion increases and from then to the 41st week the epiphyses appear as a single cap to the diaphysis. At 43 weeks some fusion of this cap to the diaphysis takes place and soon (47 weeks) cartilage is only found on the dorsal surface of the tibia. This does not disappear until the 59th week.

The proximal epiphysis of the fibula remains separate up to the 47th week. Radiographs taken at 51 weeks show the fusion of this epiphysis to be completed.

Hock Joint (Fig. 7)

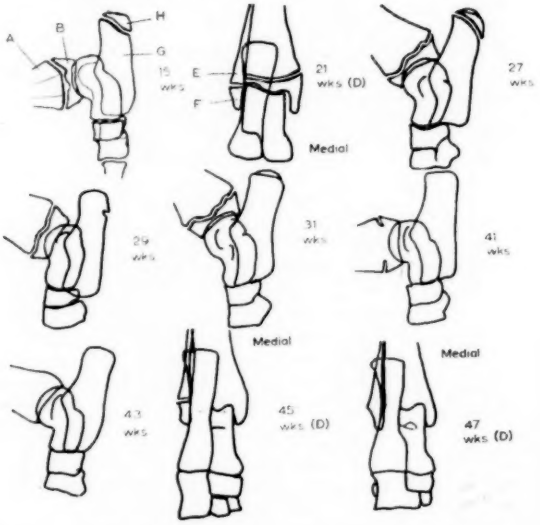

FIG. 7.—Developing hock joint. (D) Dorsal views, remainder lateral. A : tibia, diaphysis. B : tibia, distal epiphysis. E : fibula, diaphysis. F : fibula, distal epiphysis. G : fibular tarsal, body. H : fibular tarsal, epiphysis.

The tibia and fibula each have only a distal epiphysis at the 13th week. The fibular tarsal (os calcis) also has an epiphysis.

The distal epiphysis of the tibia remains separate from the shaft up to the 41st week, when fusion in the central region appears to have taken place. At 43 weeks complete fusion has occurred. The fibular distal epiphysis remains separate for slightly longer and does not appear to fuse until 47 weeks. The epiphysis of the fibular tarsal remains completely separate up to the 27th week. The next series, at the 29th week, shows some fusion to have occurred and by the 31st week the epiphysis is completely united.

The Joints of the Hind Paw (Fig. 8)

As with the corresponding parts of the fore limb the metatarsal and first two phalanges have only 1 epiphysis each. At 27 weeks most of the dogs have the epiphyses of the first and second phalanges united ; a very few have slight separation which has disappeared by the next examination. The metatarsal epiphyses unite by the 37th week.

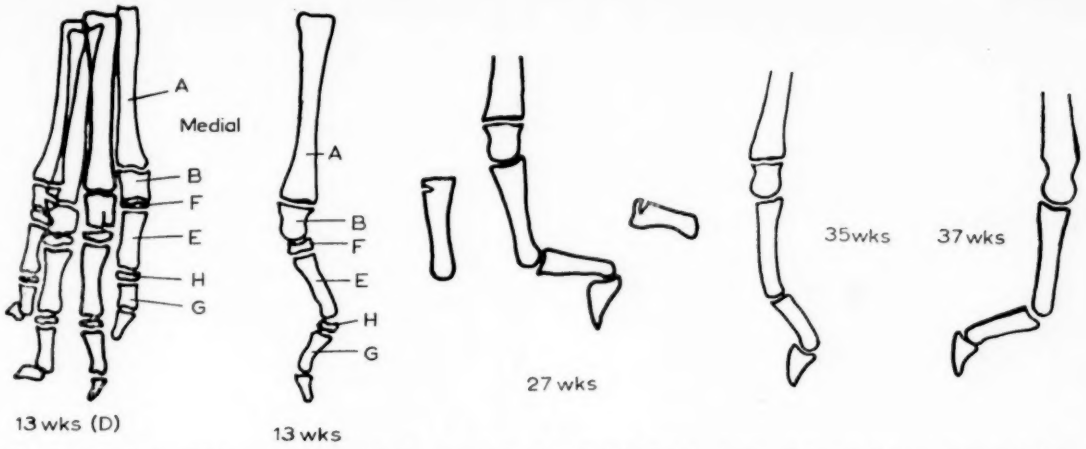

FIG. 8.—Developing hind-paw. (D) Dorsal view, remainder lateral. The first tracing is of the whole paw, the remainder of one metatarsal and digit only. A: metatarsal, diaphysis. B: metartarsal, epiphysis. E: first phalanx, diaphysis. F: first phalanx, epiphysis. G: second phalanx, diaphysis. H: second phalanx, epiphysis. It is not always possible to see the epiphysial plates of the second phalanges on a dorsal view.

Discussion

These results are presented as typical of the changes undergone by the articulations of the growing greyhound. There is, of course, some variation from the figures presented, due to sex, breeding and management. These variations are still being assessed.

The figures presented here for the greyhound are surprisingly similar to those given for Armant dogs by Seoudi (1948). The only difference is with reference to the proximal end of the radius ; this epiphysis fused completely at 9 months in his series. There is also close agreement with Hare's figures (1959), except for the same region : however, the latter worker does not give his own results and so may have been influenced by Seoudi's estimates. For the few epiphyses mentioned by the French workers there also seems to be agreement except for the distal tibial and fibular epiphyses. Their estimate for the fusion of these epiphyses is 15 to 18 months.

It should not be thought that the figures given for complete fusion of the epiphyses represent the time of cessation of longitudinal growth of a bone. The bone has ceased to grow in length at one end when the epiphysis and diaphysis unite over the smallest area, since it is only by growth of the cartilage plate that the bone elongates. Work in the Department of Veterinary Anatomy, as yet unpublished, clearly shows that in the cat, at least, cartilage remnants at the periphery of the bone can remain unossified for considerable periods after longitudinal growth has ceased.

The first time of osseous continuity is probably the more important feature and one which, by virtue of the distortion inherent in radiography, is the more difficult to appreciate. But if the cessation of longitudinal growth is to be decided by the fusion of any part of the bones then one is overlooking the possibility that growth of the cartilage plate may cease some time before it becomes ossified. In view of these facts it is surprising that ossification of the epiphysial cartilages should take place at such similar times in these various breeds and individuals.

Acknowledgments.—It is a pleasure to record our appreciation of the assistance of Messrs. Ellis, Francombe, Hawkins, Rowe and Sayer who allowed us to use their greyhounds for this work. We have also been greatly assisted by the technical staff of the Department of Veterinary Anatomy and by some of the veterinary students of the University of Bristol. We would also like to thank Professor Ottaway for the interest he has taken in this work.

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N.F.U. ANNUAL DINNER

The Annual Dinner of the National Farmers' Union was held in Grosvenor House, London, on January 25th. The retiring President, Lord Netherthorpe, was in the chair.

Amongst the senior members of the profession present were: Mr. S. L. Hignett (President of the B.V.A.), Mr. J. N. Ritchie (Chief Veterinary Officer and President of the R.C.V.S.), Dr. W. R. Wooldridge, Mr. G. N. Gould, Mr. Alan J. Wright, Mr. A. G. Beynon, Dr. R. F. Gordon, Dr. E. L. Taylor and Mr. John Anderson (Professional Secretary of the B.V.A.).

Mr. Heathcoat Amory, Chancellor of the Exchequer, proposed the toast, "Agriculture," to which the reply was made by Lord Netherthorpe. The toast of The Guests was proposed by Colonel H. J. Wilson, and the reply was made by the Rt. Hon. George Drew, Q.C., High Commissioner for Canada.

The Prophylactic and Therapeutic Activity of Metamidium and its Suramin Salt Against Trypanosomiasis in Cattle

BY

L. E. STEPHEN

Veterinary Section, West African Institute for Trypanosomiasis Research, Vom, Nigeria

SUMMARY.—1. *The chemotherapeutic and chemoprophylactic activity of a new compound—metamidium chloride—against trypanosomiasis in cattle is reported.*

2. *Metamidium chloride at a dose rate of* 10.0 *mg. per kg. is toxic for local cattle. Two oxen injected intramuscularly at this rate died.*

3. *When metamidium chloride was injected intramuscularly at* 5.0 *mg. per kg. some weight loss and initial swelling occurred but the effects were not serious. A prophylactic period of at least* 204 *days resulted.*

4. *The suramin salt of metamidium provided minimum protection periods of* 111 *and* 128 *days at* 10.0 *and* 20.0 *mg. per kg. respectively. No toxicity was evident at either of these dosage rates.*

5. *All trypanosomes breaking through the metamidium chloride or its suramin salt were* T. congolense.

TABLE II

SUMMARY OF CHEMOPROPHYLACTIC AND THERAPEUTIC ACTIVITY

Cx number	Initial prophylactic drug	Dose rate (mg. per kg.) total salt	Dose volume (ml.)	Effect on Bodyweight 1 month post dosage (kg.)	Protection period (days)	First trypanosome species seen	First re-treatment	First relapse (days)
1048	Metamidium chloride (M. & B. 4404)	5·0	26·0	− 2	204 (3)	—	—	—
1050	" "	5·0	23·0	−13	242	*T. congolense*	Metamidium chloride 0·2 mg. per kg.	24
882	" "	10·0	46·0	−27	(1)	—	—	—
936	" "	10·0	44·0	−29	(2)	—	—	—
988	Metamidium suramin salt (M. & B. 4427)	10·0	7·5	+ 3	105	*T. congolense*	Metamidium chloride 0·2 mg. per kg.	167
991	" "	10·0	9·5	− 1	111	"	" "	30
963	" "	10·0	8·5	+ 1	115	"	" "	116
966	" "	20·0	19·5	− 4	128	"	" "	93
954	" "	20·0	16·0	+ 2	137	"	" "	87
987	" "	20·0	17·0	+ 2	229	"	" "	53
923	Untreated challenged control	—	—	—	—	*T. vivax*	" "	Nil (5) (274 days)
1049	" "	—	—	—	—	"	" "	(6) 77

NOTES.—(1) Killed *in extremis* 30 days after dosing.
(2) Died 51 days after dosing.
(3) Died of piroplasmosis 204 days after dosing.

6. *Two oxen infected with* T. vivax *were treated with metamidium chloride at* 0.2 *mg. per kg. One was cured and the other relapsed* 77 *days later, but was subsequently cured by the same compound at* 0.8 *mg. per kg. It seems possible that* 0.5 *mg. per kg. as the initial treatment may cure animals infected with this species.*

INVESTIGATION of the trypanocidal activity of phenanthridinium derivatives has led to the emergence of a number of compounds possessing chemotherapeutic and chemoprophylactic activity against trypanosomes pathogenic to cattle in Africa. Recently, Wragg, Washbourn, Brown, and Hill (1958) have described the chemistry of a new trypanocide, metamidium chloride (M. & B. 4404), possessing considerable potentiality as a curative and prophylactic drug against *Trypanosoma congolense* in mice. Metamidium chloride is the *m*-amidinophenyldiazoamino-derivative of 2:7 diamino-10-ethyl-9-phenylphenanthridinium chloride (homidium chloride).

TABLE I
EXPERIMENTAL GROUPING AND DOSAGE

Number of cattle	Experimental drug	Dose rate *mg. per kg.	Dose volume ml.	Condition at time of dosing	Hair colour
2	Metamidium chloride (M. & B. 4404)	5·0	26·0 23·0	Good Fair/good	Red White
2	Metamidium chloride (M. & B. 4404)	10·0	46·0 44·0	Fair/good Fair/good	White White, black fleck
3	Metamidium-suramin salt (M. & B. 4427)	10·0	7·5 8·5 9·5	Poor Fair/good Fair/good	White White White, black fleck
3	Metamidium-suramin salt (M. & B. 4427)	20·0	17·0 19·0 19·5	Good Good Good	White White White, black fleck

* Doses refer to total drug in solution or total salt in suspension.

TABLE II
METAMIDIUM CHLORIDE AND METAMIDIUM-SURAMIN SALT IN CATTLE

First relapse (days)	Second re-treatment	Second relapse (days)	Third re-treatment	Third relapse (days)	Fourth re-treatment	Fourth relapse (days)	Weight data following re-treatment (Kg.) 1 month after first re-treatment	1 month after second re-treatment	6 months after first re-treatment
—									
24	Metamidium chloride 0·8 mg. per kg.	(4) Nil (127 days)	—	—	—	—	+11	+ 2	+38
—									
—									
167	Metamidium chloride 0·8 mg. per kg.	34	Ethidium bromide 2·0 mg. per kg.	134	—	—	− 9	+ 5	+83
30	" "	Nil (204 days)	—	—	—	—	− 7	+20	+53
116	" "	Nil (134 days)	—	—	—	—	− 6	± 0	+53
93	" "	73	Ethidium bromide 2·0 mg. per kg.	29	Berenil 2·0 mg. per kg.	Nil (122 days)	+ 8	± 0	+80
87	" "	Nil (131 days)	—	—	—	—	+37	− 5	+62
53	" "	Nil (134 days)	—	—	—	—	+17	+ 3	+49
Nil (5) 274 days	—	—	—	—	—	—	+ 3	—	+49
(6) 77	Metamidium chloride 0·8 mg. per kg.	Nil (127 days)	—	—	—	—	− 1	+ 3	+63

NOTES.—(4) Period in brackets is length of time animal kept under observation.
(5) and (6) No prophylactic drug given, therefore first re-treatment = first treatment.

The *m*-amidinophenyldiazo-amino linking group bears a structural relationship to another recently introduced curative trypanocidal drug, "Berenil," *p*-amidinophenyldiazo-amino-*p*-benzamidine.

A report is now given of the therapeutic and prophylactic activity of the chloride of metamidium and the suramin salt (M. & B. 4427) (Williamson & Desowitz, 1956) against West African strains of *T. vivax* and *T. congolense* in cattle.

The trial* was arranged to take place at this Institute by the Chemotherapy Panel of the Tsetse Fly and Trypanosomiasis Committee of H.M. Colonial Office.

Materials and Methods

Drugs. (a) Metamidium chloride (M. & B. 4404) is presented as a dry powder. It was made up as a 4 per cent. w/v solution in sterile distilled water.

(b) The suramin salt of metamidium chloride (M. & B. 4427) is presented as a very sparingly soluble dry powder. It was made up as a 20 per cent. w/v suspension in sterile distilled water.

Injection Site. The drugs were injected intramuscularly into the muscles overlying the wing of the atlas vertebra.

Cattle. Twelve White Fulani Zebu bulls were purchased from the Federal Department of Veterinary Research, Vom. They had all been immunised against rinderpest and some had been used recently in bovine pleuropneumonia experiments. Their condition ranged from poor to good.

Fly Challenge. The fly challenge, which is routine at this Institute, was employed. Full details of this challenge have been published (Stephen, 1958). Essentially it consists of feeding batches of 100 wild-caught *Glossina morsitans* for 3 consecutive days, at intervals of 10 to 14 days, on the treated cattle. The flies are kept in Bruce boxes. Before entering the cattle challenge they are fed daily for 10 or 11 days on sheep showing a high trypanosome parasitaemia, in order to increase the natural infection rate. Fly dissection data has shown that the predominant trypanosome in this challenge is *T. vivax*.

Routine Examinations. The rectal temperature of each animal was taken daily between 7 and 9 a.m. Wet blood films were examined for trypanosomes for 14 days before dosing, to ensure the absence of a pre-dosage infection, and thereafter 6 days a week for the duration of the experiment. Each animal was weighed 4 times before dosing and then at weekly intervals, and was examined clinically by the author at least 3 times a week. At 10- to 14-day periods blood was examined by techniques already reported (Stephen, 1958; Stephen & Williamson, 1958), as follows:—

(a) Erythrocyte count.
(b) Packed Cell Volume (Haematocrit).
(c) Haemoglobin.
(d) Serum Bilirubin.
(e) Serum Non-protein Nitrogen.
(f) Plasma Specific Gravity.
(g) Formol Gel Test.

* The cattle and drugs were provided by Messrs. May and Baker Ltd., Dagenham, Essex.

Controls. Two oxen were used as controls, initially for comparison of weight and blood values; later they were exposed to the fly challenge to confirm its effectiveness. When they became infected, each ox was treated with metamidium chloride at 0.2 mg. per kg. bodyweight.

Husbandry. Grazing was poor during the first 5 months of the trial and supplementary feeding was provided as follows: atcha straw (*Digitaria exilis*), approximately 8 lb. per head per day; groundnut cake, approximately 1.5 lb. per head per day; rock salt (local "Kanwa"), and a balanced mineral mix *ad libitum*. The animals were housed in cattle sheds from sunset to sunrise.

Results

A. *Metamidium Chloride (M. & B. 4404)*

Toxicity. A dosage rate of 10.0 mg. per kg. bodyweight was lethal. Within 30 minutes 2 oxen dosed at this level showed lachrymation, salivation, tachycardia, jugular pulse, polypnoea, staggering, and a tendency to walk backwards. One animal collapsed. Severe, oedematous swellings were observed within 24 hours over the entire side of the neck and jaw, and in the intermandibular space. One beast was killed, *in extremis*, 30 days later, and the other died 51 days later, having lost 27 and 55 kg. bodyweight respectively.

At autopsy there were no gross pathological changes in these animals, except at the site of inoculation. Haematological and clinical chemical examinations were not significantly altered. Histopathological examination similarly gave no apparent indication of the cause of death (Beveridge, 1959).

The 2 oxen dosed at 5.0 mg. per kg. developed moderately severe swellings around the injection site, which persisted for about 6 weeks. There was also some stiffness of the atlanto-occipital joint, but this did not affect grazing. Both animals lost some weight during the first month after dosing, but this was not serious.

Therapeutic Activity. The two control oxen were subsequently placed in the routine fly challenge and became infected with *T. vivax*, 10 and 11 days respectively after the first flies had fed. They were treated 6 and 5 days later respectively with metamidium chloride injected intramuscularly into the triceps, at a dosage rate of 0.2 mg. per kg., employing a 1.0 per cent. w/v solution. One beast became negative and did not relapse during an observation period of 9 months; the other relapsed 77 days after treatment. The relapse infection was very scanty—trypanosomes were seen in wet preparations on 4 occasions only during the 144 days of observation from relapse to re-treatment, and the infection did not seem to affect the health of the animal adversely, in fact it gained 74 kg. bodyweight during the period. The animal was then re-treated with metamidium chloride at 0.8 mg. per kg. and this appeared to cure the infection (observation period, 127 days).

During the course of other experiments at this Institute, metamidium chloride given intramuscularly at a dose rate of 0.8 mg. per kg. has cured *T. congolense* infections which were resistant to homidium bromide at 1.0 mg. per kg. and antrycide dimethyl

sulphate at 5.0 mg. per kg. Since these trypanosomes had broken through suramin salts of "Prothidium" and R.D.2902 (2-amino-7-(2′-amino-1′:6′-dimethylpyrimidyl - 4′ - amino) - 9 - phenyl-10-ethylphenanthridinium dimethanesulphonate) administered at 5.0 mg. per kg., they were presumably resistant to some extent to these drugs also. These observations will be published in more detail at a later date.

Prophylactic Activity. The 2 beasts which received metamidium chloride at 5.0 mg. per kg. resisted infection in the routine fly challenge for 204 and 242 days respectively. The former never became infected but died of piroplasmosis. The other developed an infection with *T. congolense* at the end of the protection period. This breakthrough infection was treated 4 days later with metamidium chloride at 0.2 mg. per kg. and a relapse occurred 24 days later. This relapse was then treated with the same drug at 0.8 mg. per kg. No relapse occurred during the observation period of 127 days.

B. *Metamidium-Suramin Salt (M. & B. 4427)*

Toxicity. There was no evidence of systemic toxicity in any of the cattle treated at 10.0 or 20.0 mg. per kg. The immediate post-injection swellings were very slight in all cases and most of them rapidly resolved. However, one animal in each dose group developed a slow-growing lump which at 6 months was about 5 by 5 inches and 2 to 3 inches prominent. These swellings persisted throughout the observation period, but did not appear to inconvenience the animals in any way.

Prophylactic Activity. The 3 oxen which received the metamidium-suramin salt at 10.0 mg. per kg. were protected for 105, 111, and 115 days respectively. Treatment of the break-through infections, with metamidium chloride at 0.2 mg. per kg., resulted in relapses 167, 30, and 116 days later respectively. These relapse infections were then treated with metamidium chloride again at 0.8 mg. per kg. Two beasts were cured by this treatment (observation period 204 and 134 days), but the third relapsed 34 days later and it was then treated with homidium bromide at 2.0 mg. per kg. A third relapse occurred 134 days after this treatment. On that day *T. congolense* was seen in wet films on 2 separate occasions, and also in Giemsa-stained thin films, but trypanosomes have not re-appeared in the peripheral blood during the 120 days since this relapse.

An attempt was made to recover the trypanosome by xenodiagnosis, *i.e.* by allowing 100 clean *G. morsitans* to feed on the animal for 14 days and then on a clean ox for 11 days. The clean animal never became infected, and no infection was found when 40 of the flies were dissected at the end of the experiment. When 300 ml. of citrated blood was sub-inoculated into a clean ox, however, the recipient became infected with *T. congolense* 11 days later. The recipient did not become clinically ill during an observation period of 80 days, although a moderate parasitaemia persisted during that time.

The treatment of the breakthrough infection with metamidium chloride at 0.2 mg. per kg. resulted in slight weight loss in all 3 of the animals in this group during the month following treatment. Thereafter, despite relapses and subsequent treatment, quite satisfactory weight gains were recorded; for example, after 6 months they had gained 83, 53, and 53 kg. respectively.

The 3 oxen dosed with the metamidium-suramin salt at 20.0 mg. per kg. were protected for 128, 137, and 229 days respectively. They all relapsed when treated with metamidium chloride at 0.2 mg. per kg. in 93, 87, and 53 days. These relapse infections were then treated with the same compound at 0.8 mg. per kg. Two were apparently cured by this treatment (observation period 134 and 131 days) but the third relapsed after 73 days. Treatment of the third animal with homidium bromide at 2.0 mg. per kg. resulted in a third relapse 29 days later. It was then decided to examine the effect of "Berenil" on this infection. It will be recalled that metamidium chloride bears some structural similarity to this drug. Accordingly, the animal was injected intramuscularly with "Berenil" at a dose rate of 2.0 mg. per kg. employing a 7 per cent. w/v soluton. Wet films were negative during an observation period of 122 days after this treatment.

None of these cattle lost weight after treatment with metamidium chloride at 0.2 mg. per kg. although one lost 5 kg. after treatment with this compound at 0.8 mg. per kg.; however, 6 months after the first retreatment they had gained 80, 62, and 49 kg. respectively.

All trypanosomes which broke through metamidium chloride or metamidium-suramin salt were identified as *T. congolense*.

These results are summarised in Table II. No adverse systemic effects were observed in the biochemical examinations performed, nor was the blood picture altered in any significant manner.

Discussion

Metamidium chloride is very toxic when injected intramuscularly in West African White Fulani Zebu bulls at 10.0 mg. per kg. bodyweight, but the same drug at 5.0 mg. per kg., although it causes some initial weight loss and swelling at the injection site, is reasonably well tolerated. The results of this experiment indicate that a prophylactic period of 6 to 7 months may be expected following a single intramuscular injection of metamidium chloride at a dose rate of 5.0 mg. per kg. However, field trials on a large scale must be made before this period can be accepted with any degree of certainty.

Employing the metamidium-suramin salt at 10.0 mg. per kg. a protection period of the order of 3 months was obtained. Doubling this dose extended the period of protection only for about 1 month.

Therapeutically, metamidium chloride is active against *T. vivax* and *T. congolense*. It appears that a dose of 0.8 mg. per kg. is sufficient to cure infections due to these organisms provided that they have had no previous contact with metamidium chloride or its suramin salt. It is possible that this dose might be reduced to 0.5 mg. per kg. in such infections, but this possibility has not been experimentally examined. However, cure of break-through infections previously in contact with the metamidium-

suramin salt cannot be reliably effected with metamidium chloride even at 0.8 mg. per kg.

Homidium bromide also appears to be of doubtful value as a curative drug against trypanosomes resistant to metamidium chloride and its suramin salt. This is not surprising as metamidium chloride, chemically, can be considered as a substituted homidium. Unexpectedly, however, "Berenil" seems to possess considerable activity against organisms resistant to the metamidium salts *and* homidium bromide. This may be related to the steric difference between the *p*-isomer ("Berenil") and its *m*-isomeric moiety in metamidium chloride.

Metamidium chloride has shown considerable activity in other experiments against strains of *T. congolense* resistant to homidium bromide, "Prothidium" and the closely related R.D.2902, despite its chemical similarity to these phenanthridinium compounds. It thus appears that its activity against phenanthridinium-resistant trypanosomes is due to the amidinophenyldiazoamino portion of the molecule.

The writer believes that prophylactic regimes in cattle should be organised so that re-treatment occurs before the minimum protection period expires. Average protection periods and the range of time over which breakthroughs occur should be used as as a means of estimating the probable *minimum* protection period. If animals are not re-treated until after this minimum protection period, then a drug of different chemical composition and with high therapeutic activity, particularly against strains resistant to the first drug, should be administered before re-treatment with the original prophylactic. Failure to adhere to a treatment plan of this nature will soon result in a rapid decline in the effectiveness of the original drug because of the production of drug-resistant trypanosomes.

In many breakthrough and relapse infections trypanosomes appear in the peripheral blood only in very small numbers and at infrequent intervals. Cattle infected with such organisms often show little evidence of disease and they may even thrive. It seems possible that pathogenicity has been diminished and therefore the effects, pathogenic or otherwise, of such trypanosomes upon cattle that have never received a prophylactic drug are now being investigated.

The failure to recover the trypanosome resistant to 0.8 mg. per kg. of metamidium chloride and 2.0 mg. per kg. of homidium bromide by the technique of xenodiagnosis, and the fact that this organism did not cause clinical illness in a clean animal infected by subinoculation of blood is of considerable interest. Experiments are in progress to determine whether (a) drug-resistant trypanosomes lose some of their ability to develop cyclically in the fly, and (b) such organisms are less pathogenic than they were before they became drug-resistant.

The criterion of a cure is of great importance in conducting a drug trial. It will be seen from Table I that one beast relapsed 167 days after treatment. Post-treatment observations should probably be continued for a period of 6 months to be certain that a cure has been effected. Unfortunately this is seldom possible because of the cost or physical difficulties of maintaining cattle for so long a period.

Experiments are in progress to examine the prophylactic activity of metamidium chloride at lower dose rates, and also to investigate its therapeutic activity against *T. simiae*. A different particle size of the metamidium-suramin salt will also be examined, as the preparation used in this experiment may have been too insoluble.

"Berenil" will be used to treat all trypanosomes breaking through metamidium chloride or its salts in these experiments in order to assess its therapeutic value in such cases in larger numbers of animals.

Acknowledgments.—The author wishes to thank Mr. K. E. Broomfield for his valuable assistance during this trial. Thanks are also due to Dr. J. Williamson for advice on the chemical constitution of the drugs used; to Dr. K. C. Willett and Mr. A. R. Gray for critically reading the manuscript; and to the African technicians and herdsmen for diligently carrying out their duties.

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MR. THOMAS WILSON

At the recent quarterly meeting of the council of the Royal College of Veterinary Surgeons, the treasurer, Mr. Harry Sumner, made reference to the late Mr. Thomas Wilson of Nantwich, who had been a member of the College council from 1938 to 1945. Mr. Sumner said:—

"I should like to refer to the passing of Mr. Thomas Wilson. Mr. Wilson was a notable and outstanding practitioner in Cheshire and one who, in spite of the calls of his practice, found time to serve the profession to the very limit of his abilities. He was a member of this Council for some time and that period marked him out as one of those valuable servants of the profession whose work was notable and more outstanding in committee than in the full public view of council proceedings. He was a man who took a lively and an important part in the proceedings of the Lancashire Division and in addition was a very strong supporter and took a very active part in the proceedings of the National Veterinary Benevolent and Mutual Defence Society where his wisdom helped us to come to some very wise and proper decisions.

Personally he was extraordinarily kindly, and possibly his kindness had its greatest outlet in the consideration and help he gave to the younger members of the profession. He was a great friend of us all, we feel his passing, and I should like some expression of sympathy to go to his family."

The Effect of Chlorpromazine on the Cardiovascular System of the Conscious Horse

BY

L. W. HALL

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IN some horses the intravenous or intramuscular injection of chlorpromazine may induce a state in which the animal gradually sinks down on its haunches and then leaps forward and manifests great excitement or panic. This excitement or panic gradually subsides and the whole sequence of sinking down, plunging forwards and excitement is repeated over and over again until the effects of the drug are no longer apparent. Most horses do not behave in this way following the administration of chlorpromazine but there is no way of predicting when the "panic reaction" will be encountered. Owen and Neal (1957) pointed out that chlorpromazine may induce a state of hypotension in horses (Martin & Beck, 1956) and the descriptive literature supplied by the manufacturers of the drug suggests that this effect may be responsible for the "panic reaction." It is, therefore, of interest to record some observations which were made in the course of a recent investigation into the pharmacological effects of the phenothiazine derivatives, for they indicate that in horses the excitement or panic is not, in fact, related to any hypotensive effect of chlorpromazine.

Experimental Procedure

It was found that the intravenous injection of 0·5 mg. per kg. of chlorpromazine hydrochloride regularly produced a state of excitement or panic in a Welsh Pony mare which had been provided surgically with an exteriorised carotid artery from which continuous blood pressure recordings could be made.

The blood pressure was recorded on a direct writing electromanometer (Cambridge Instrument Co. Ltd.) from a catheter attached to a needle introduced into the exteriorised carotid artery. Drugs were injected into the jugular vein.

Results

After obtaining a control record (Fig. 1), 0·5 mg. per kg. of chlorpromazine hydrochloride was injected intravenously and the effect of this drug on the blood pressure observed. The blood pressure increased from the control value of 114/64 (*i.e.* systolic pressure 114 mm. of mercury, diastolic pressure 64 mm. of mercury) until after 5 minutes it was 120/70 while the pulse rate increased from 60 to 180 per minute. Seven minutes after the injection of chlorpromazine the pony commenced to exhibit the reaction described above and the relevant portion of the blood pressure tracing is shown in Fig. 2. The violent waves seen at the end of this portion of the tracing were caused by movement of the recording catheter as the animal plunged forward. The blood pressure at this time was about 120/70 and the pulse rate about 180 per minute. One hour later the blood pressure was unchanged (120/70) but the pulse rate had fallen to 120 per minute and the pony was no longer showing the "panic" reaction.

Sixteen days later the pony was given 3·0 mg. per kg. of a ganglionic blocking agent, hexamethonium bromide. Following the administration of the hexamethonium the blood pressure fell to as low as 64/25 and the pulse rate increased from a control value of 64 to 84 per minute (Fig. 3) while the respiratory rate increased from 12 to 40 per minute. The depth of respiration was also noticeably greater but at no time did the pony manifest any sign of the "panic"

WELSH PONY MARE: BLOOD PRESSURE TRACINGS
(Paper speeds shown alongside Tracings)

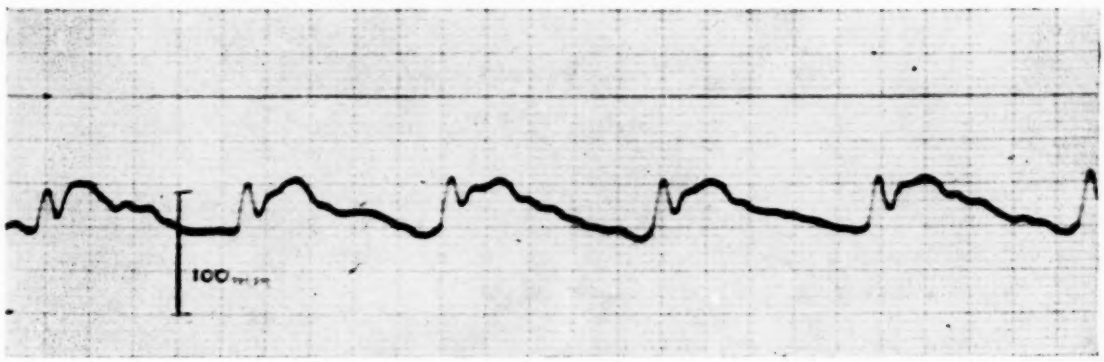

FIG. 1.—Normal resting state. 2.5 cm./sec.

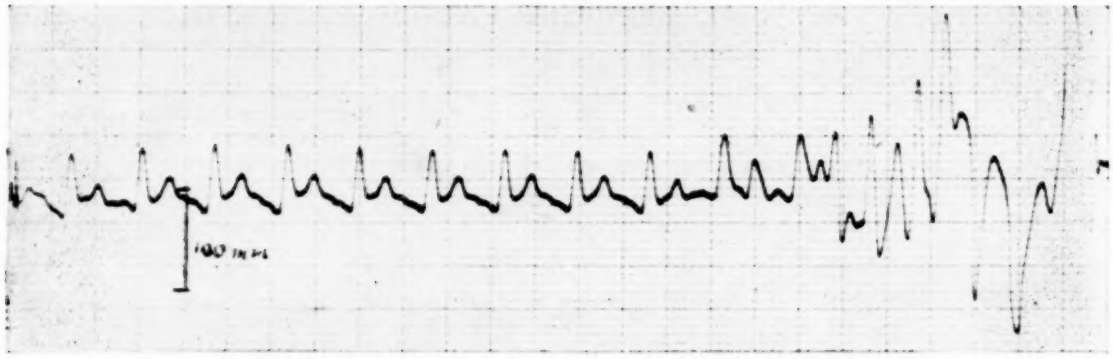

FIG. 2.—Under the influence of chlorpromazine (0.5 mg. per kg. intravenously). Very excited, jumping and plunging forward. Note the artefacts caused by disturbance of the catheter. 2.5 cm./sec.

reaction. (The artefacts in the tracing shown were due to movements of the head and neck disturbing the catheter).

Discussion

The results of these 2 experiments show that in this particular animal the " panic " reaction induced by the intravenous injection of 0·5 mg. per kg. of chlorpromazine was not associated with a state of hypotension. Furthermore, the deliberate induction of hypotension with hexamethonium did not induce a state of " panic." These findings suggest that some other explanation for the " panic " reaction must be sought and it is possible that this reaction may be due to a subjective feeling of distress caused by the severe tachycardia, or the reaction may be unassociated with any cardiovascular effect of the drug.

Owen and Neal (1957) also suggested that the reaction may be provoked by a feeling of weakness in the limb muscles, and in man, Dobkin, Gilbert and Lamoureux (1954) Lehman and Hanrahan (1954) and Anton-Stephens (1954) have recorded muscle weakness induced by the administration of chlorpromazine. It is perhaps significant that horses sink slowly down on their haunches before plunging forward and becoming excited. However, it must be noted also that for some, as yet completely unknown reason, chlorpromazine produces what have been described as " paradoxical responses " in other animals. In man, most experienced observers have encountered occasional cases in which the response shown was different from, and in some cases the exact opposite to, the sedation usually produced by chlorpromazine. Similar observations have also been made on rare occasions in dogs and cats (Hall, 1955) and it is possible that these odd paradoxical responses are encountered much more frequently in horses than in other animals, so that only in horses is the drug particularly unreliable as a sedative.

Further investigation into the cause of the " panic reaction " is obviously desirable, for until it is known what particular pharmacological action of chlorpromazine is responsible for its occurrence it will be impossible to assess from a consideration of the known pharmacological effects whether any particular phenothiazine derivative will be useful in horse practice.

(*Concluded overleaf*)

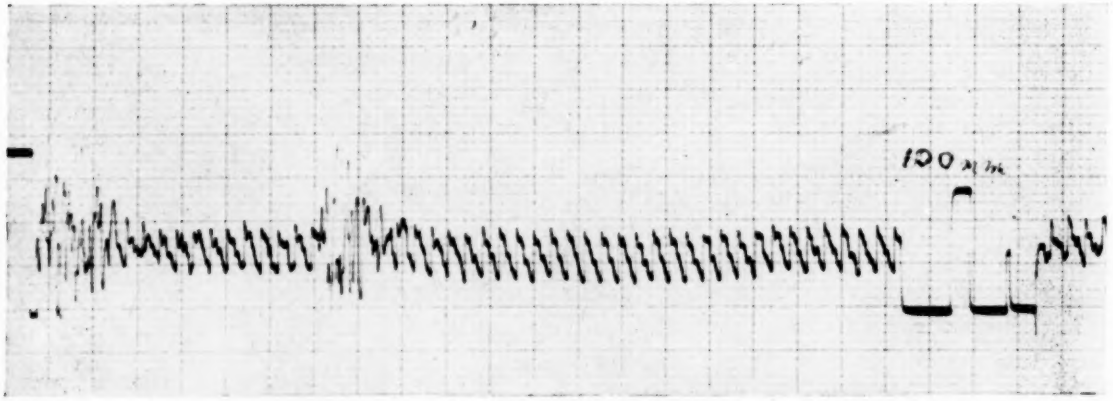

FIG. 3.—Hypotension induced with hexamethonium bromide (3.0 mg. per kg. intravenously). No excitement. 0.25 cm./sec.

Lack of Pathogenicity of Rous 1 Virus for Humans: A Long-Term Experiment

BY

J. G. CARR

British Empire Cancer Campaign Unit, Poultry Research Centre, Edinburgh, 9

THE incidence of fowl leucosis is very high in commerical flocks in this country (Blaxland, 1956) and even higher in the U.S.A. (U.S. Dept. Agric. Circular, 1955). A considerable proportion of the survivors are now regarded as carriers of the causative virus(es) and infection through the eggs of such birds is believed to be an important mode of transmission (Cottral, Burmester & Waters, 1954; Burmester, Gentry & Waters, 1955). Thus, the question of the reaction of these viruses with humans is being increasingly raised. Though such reaction is unlikely, no direct evidence seems to be reported in the literature, and therefore a deliberate experiment with a survival at more than 20 years seems worth recording to aid in quietening such apprehensions.

In April, 1939, 2 injections of 0.2 ml. of purified Rous 1 virus suspension as prepared by the method of Amies and Carr (1939) were injected subcutaneously into the left forearm, and this was repeated with another preparation 2 weeks later. Simultaneous titration on chicks showed that the preparations contained about 100 and 1,000 infective doses *per inoculum*, respectively. A slight redness and irritation was produced which disappeared in about 48 hours, and nothing further has resulted to date.

Rous and Murphy (1914) failed to infect adults of any laboratory animals with the Rous 1 virus, though all avian embryos seemed susceptible (Murphy & Rous, 1912). Later Andrewes (1932) successfully infected pheasants with this virus, and Duran-Reynals (1913) showed that very young birds of other species were often susceptible. The young rat reacts to the virus (Svet-Moldavsky, 1957), while tumour cells will survive for 12 days in the adult rat (Fujinami & Hatano, 1929) and grow in the mouse after blockade of the reticulo-endothelial system (Roskin, 1927). Other avian viruses are less selective (*e.g.* Fujinami (1930) showed that his virus will grow in ducks, quails, pigeons, etc.) and so are mutants of the Rous 1 virus derived from inoculation of insusceptible hosts such as the duckling (Duran-Reynals, 1947) or guinea-pig eye (Shrigley, Green & Duran-Reynals, 1945). These mutants may need many months before the carcinogenic action becomes apparent.

Cooking would, of course, destroy the virus, and in any case it was long ago shown by Rous and his colleagues (1912) that susceptible birds can be fed on the virus with impunity.

Acknowledgment.—All expenses in connexion with this experiment were borne by the British Empire Cancer Campaign.

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The Effect of Chlorpromazine, etc.—*Concluded.*

Acknowledgments.—Financial assistance and generous supplies of chlorpromazine hydrochloride and hexamethonium bromide were provided by May and Baker Ltd.

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MR. ALLEN BAKER

Allen Baker, M.R.C.V.S., died suddenly at his residence, Lismacue, Bansha, Co. Tipperary, on December 20th, 1959. The son of Mr. Charles Conyers Massy Baker, he was born in London in 1881, first went to Lismacue in 1896, and qualified in Dublin in 1904. Allen Baker won the first Gold Medal at Dublin Veterinary College in 1904 for securing the highest number of marks in the final examination. It is also of interest to record that he was the first Irish veterinary surgeon to sign the register.

On the death of his father in 1905, he took over the management of Lismacue where he established a stud of the highest standard. How successful he was as a breeder may be judged from the fact that from 1938 to 1959 he bred the winners of 395 races to the value of £114,423. He was a man of varied country pursuits, and his love of flowers and of horses were two of his greatest interests in life.

The news of his death came as a profound shock to his many friends and acquaintances. We extend our sympathy to his wife and three children.

News and Comment

B.V.A. ANNUAL CONGRESS SCIENTIFIC EXHIBITION

It is hoped to organise a scientific exhibition in connexion with the 78th Annual Congress, to be held in Glasgow from August 29th to September 3rd, 1960. All members of the veterinary profession, and organisations concerned with veterinary science in all its disciplines, are cordially invited to contribute.

Exhibits may take the form of charts, diagrams, drawings, models, photographic prints or transparencies, radiographic plates, microscopic preparations, anatomical or pathological specimens, apparatus, techniques, etc. All exhibits must be accompanied by suitable legends or explanations, and bear the names and places of work of the exhibitors, and will have to be assembled by those submitting them not later than Sunday, August 28th.

Intending contributors are asked to get in touch with Dr. Alastair Greig, University Veterinary Hospital, Bearsden Road, Bearsden, Glasgow, as soon as possible (and in any case not later than April 30th), and to indicate the general nature of the exhibit they propose and the approximate dimensions of the bench and/or wall space they would require. A certain amount of apparatus, *e.g.* microscopes, will be available for the use of exhibitors, but it is not expected that the supply of viewers for transparencies, slides, and radiographic plates will be sufficient to meet the demand. It would be very helpful if intending exhibitors were to intimate their requirements as early as possible, particularly if these are of a specialised nature.

A.R.C. VETERINARY RESEARCH FELLOWSHIPS

The Agricultural Research Council is offering two Veterinary Research Fellowships, tenable for 3 years, to provide opportunities for suitably qualified veterinarians to undertake research. Candidates must be British subjects and hold a registrable veterinary qualification, and should have been engaged in research for not less than 3 years since qualifying.

The Council is also continuing to offer postgraduate Research Studentships to holders of veterinary qualifications, and a small number of veterinary Training Grants to enable honours graduates in one of the basic sciences to read for a veterinary qualification. Applications for these awards should reach the Council by March 15th, 1960.

Further details of the above awards, together with information about openings in agricultural research and in advisory and investigational work, are obtainable from the secretary, Agricultural Research Council, Cunard Building, 15, Regent Street, London, S.W.1.

B.S.A.V.A. THIRD ANNUAL CONGRESS

This congress will be held at the Washington Hotel, Curzon Street, London, W.1, on March 18th, 19th, and 20th. The most important innovation is the introduction of closed-circuit colour television, arranged through the courtesy of a leading pharmaceutical house (Smith, Kline and French). Telecasts will take place on the afternoons of Friday and Saturday and will demonstrate various surgical procedures. The emphasis will be on practical interest to the small-animal practitioner, rather than the complex-type of surgical procedure more fitted to the operating theatre of a veterinary school. During each session the surgeon will be able to answer questions from the audience and the procedures will be discussed by a special panel sitting in front of another television camera. Among the items to be shown on television are blood collection and transfusion, removal of canine and carnassial teeth from the dog, total mastectomy, and an orthopaedic procedure.

Apart from the television sessions, papers will be read on the problems of cat practice, regulations concerning the export of dogs, and there will be a symposium on the subject of internal parasites in the dog.

Owing to the increased number of applicants for space in the trade exhibition, this has now been moved to a larger room and 31 trade exhibitors will attend. The exhibition will be open throughout the Congress. In addition, a small scientific exhibition, entirely photographic, will be mounted in the main Congress room covering subjects of interest to the small-animal practitioner; among these will be fungal infections, a demonstration of canine internal parasites and a number of general clinical photographs.

The Ladies' Committee have arranged a number of events for visitors' wives. These will include a special visit to Norman Hartnell's dress salon and a demonstration of flower arrangement by a representative from Constance Spry. On the Saturday evening, there will be a dinner and dance. Members of the B.S.A.V.A. will receive application forms and full details by post but non-members should apply for these to the honorary treasurer, Mr. J. S. J. Lauder, The Priory, London Road, Brighton. Members will be admitted for a registration fee of one guinea and facilities will be available for non-members to join the Association at the Congress. Visitors should make their own hotel arrangements either with the Washington Hotel or any other hotel of their choosing. Over 300 people attended the last Congress and it is advisable, therefore, to book hotel accommodation at the Washington as early as possible.

PERSONAL

Engagement

PARRIS—PONSFORD.—The engagement is announced between John Christopher Parris, B.V.SC., M.R.C.V.S., of 98, Monnow Street, Monmouth, and Frances June Ponsford, S.R.N., of Exeter, Devon.

Marriages

BROUGH—MACKENZIE.—At Inverness, on January 5th, 1960, Robert J. Brough, B.V.M.S., M.R.C.V.S., of Glasgow, to Cuprenia Mackenzie of Portree, Isle of Skye.

COLLINS—BONSER.—On January 16th, 1960, at Westbury Park Methodist Church, Bristol, Lt. George Henry Collins, B.V.SC., M.R.C.V.S., of 53, Upper Cranbrook Road, Bristol, to Dorothy Muriel Bonser of 39, George Street, Warsop, Mansfield, Notts.

Birth

POOLE.—On December 24th, 1959, at Market Rasen, Lincs., to Rhoda (*née* Guilford), wife of A. Poole, B.SC., M.R.C.V.S., a son, Graham Scott.

COMING EVENTS

February

3rd (Wed.) Meeting of the Southern Counties Veterinary Society at the Royal Hotel, Winchester, 7.30 p.m.

4th (Thurs.). Meeting of the Central Veterinary Society at the Royal Veterinary College, Camden Town, N.W.1, 6 p.m.

5th (Fri.) Annual Ball of the Royal (Dick) School of Veterinary Studies in the Assembly Rooms, George Street, Edinburgh, 9 p.m.

Meeting of the Shropshire Veterinary Club at the Grapes Hotel, Bicton Heath, Shrewsbury, 8 p.m.

Meeting of the West of Scotland Division at the University Veterinary Hospital, Bearsden Road, Bearsden, 2.30 p.m.

16th (Tues.). Keith Entwistle Memorial Lecture in the Old Library, Pembroke College, Cambridge, 8.15 p.m.

Annual General Meeting of the Eastern Counties Veterinary Society at the Bell Hotel, Norwich, 10.30 a.m.

17th (Wed.). Annual General Meeting of the South Eastern Veterinary Association at the Royal Star Hotel, Maidstone, 2.30 p.m.

Annual Joint Meeting of the Sussex Veterinary Society with the Brighton and Mid-Sussex Division, B.M.A., at the Dudley Hotel, Hove, 8 p.m.

19th (Fri.). Buffet Dance of the V.V.B.F. at the Talardy Hotel, St. Asaph, North Wales, 8.30 p.m.

24th (Wed.). Annual General Meeting of the Lancashire Veterinary Association in Liverpool, 2.30 p.m.

26th (Fri.). Annual Ball of the Liverpool University Veterinary Society, 8 p.m.–2 a.m.

March

3rd (Thurs.). Meeting of the Society for the Study of Animal Breeding at the Royal Society of Medicine, 1, Wimpole Street, London, W.1, 11.30 a.m.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Dumfries. Croftheads Farm, Annan (Jan. 22).
Hants. Manor Farm, Grateley, Andover (Jan. 22).
Worcs. Stable, Mill House, Kempsey (Jan. 20).

Fowl Pest

Cambs. Hazeldene, High Road, Gorefield, Wisbech; Manor Farm, Towlmere, Royston (Jan. 19); Potatoe Drove, Marshland St. James, Emneth, Wisbech; Ranch Farm, Marshland St. James, Wisbech (Jan. 22).

Essex. Hope Cottage, Arkenden Road, Clavering, Saffron Walden (Jan. 21); Kiln Farm, River View, Pitsea, Basildon (Jan. 23).

Hants. Tunlands Farm, House, Over Wallop, Stockbridge (Jan. 19); Ringwold House, Middle Wallop, Stockbridge (Jan. 21).

Hunts. Cambridge Road Allotments, Godmanchester (Jan. 21); Cow Lane Corner, Godmanchester (Jan. 22).

Lincs. Wigg Lane, Chapel St. Leonards, Skegness (Jan. 24).

Norfolk. Bingles Turn, Hevingham, Norwich; Stone Cottages, West Harling Road, East Harling, Norwich; Bromley House, Stow Bedon, Attleborough; Spring Cottage Farm, Great Dunham, King's Lynn; Topps Hill Farm, Topps Hill Lane, Thorpe Market, Norwich; Lodge Farm, Barney, Fakenham; Mill Lane, Hockwold, Thetford; White House, London Road, Attleborough; High Green, Brooke, Norwich (Jan. 19); Bottom End Farm, Dunston, Norwich; Fairfield, Newton, St. Faith, Norwich; 26, Upper Street, Gissing, Diss; Swans Farm, Tuttington, Norwich; Hillside Farm, Brooke, Norwich; Poplar Farm, Hickling, Norwich; The Pope, Ram Lane, Tivetshall St. Mary, Norwich; Peewit Farm, Wood Dalling, Norwich; Vernon Bungalow, Bradfield Road, Southrepps, Norwich (Jan. 20); Post Office Stores, Weybread, Diss; Mere Farm, Stow Bedon, Attleborough; 6, Tottington Lane, Roydon, Diss; Gravel Pit Farm, Bergh Apton, Norwich; Wash Farm, Banham, Norwich; Mill House, Mill Lane, Hockwold, Thetford (Jan. 21); Church Road, West Dereham, King's Lynn; Street Farm, Bergh Apton, Norwich; Beech House, Bergh Apton, Norwich; Green Farm, Deopham, Wymondham; Foundry Hill Farm, Wood Dalling, Norwich; Foundry Hill, Wood Dalling, Norwich; Nursery Lane, Hockwold, Thetford; Manor Farm, Fleggburgh, Great Yarmouth; Hevingham Ave. Farm, Hevingham, Norwich; Kemps Lane, Hockwold, Thetford (Jan. 22); Lodge Farm, Hainford, Norwich; Lime Kiln Farm, Heydon, Norwich; Poplar Farm, Sisland, Loddon, Norwich; South Street, Hockwold, Thetford; Kimberley House, Hall Lane, Hingham, Norwich; Thorpe Hall, Mendham, Harleston; The Bungalow, Mill Corner, Hingham, Norwich; Town Farm, Metfield, Harleston (Jan. 23); 4, Red Lion Street, Aylsham, Norwich; White Lodge Farm, Hingham, Norwich (Jan. 24).

Suffolk. 79, Station Road, Lakenheath, Brandon; 81, Station Road, Lakenheath, Brandon; 57, Station Road, Lakenheath, Brandon; 25, Station Road, Lakenheath, Brandon; 1, Station Road, Lakenheath, Brandon (Jan. 19); Home Farm, Weeting, Brandon (Jan. 20); Osborne House, Ilketshall St. Lawrence Beccles; Ouse Bungalow, Lakenheath, Brandon (Jan. 21); The Street, Badwell Ash, Bury St. Edmunds (Jan. 22); Green Lane, Fenningham, Stowmarket (Jan. 23); Cattle Farm, Yaxley, Eye (Jan. 24).

Yorks. Maythorn Farm, Leyburn (Jan. 19); Low Swetton Farm, Dallowgill, Ripon; Fox Holme, Park Square, Masham, Ripon (Jan. 20).

Swine Fever

Cornwall. Willow Green Farm, Three Milestone, Truro (Jan. 19); Bosvargus St. Just, Penzance (Jan. 20); Carnkie Farm, Wendron, Helston (Jan. 25).

Cumbs. Drawdykes Castle Farm, Carlisle (Jan. 25).

Devon. Sheafhayne Farm, Yarcombe, Honiton (Jan. 22).

Hunts. Marsh Lane, Hemingford Grey (Jan. 19); The Hollies, Colne (Jan. 22).

Lancs. Stanley House Farm, Staining, Poulton le Fylde, Blackpool (Jan. 20).

Leics Shoby House Farm, Shoby, Melton Mowbray (Jan. 25).

Norfolk. Mill Farm, Bacton Road, North Walsham (Jan. 20); Church Farm, Cawston, Norwich (Jan. 21); Middle Farm and Carslough Farm, Hethel, Wymondham (Jan. 22); Church Farm, Wreningham, Norwich (Jan. 25).

Sussex. Down Farm, Roack Lane, Guestling Green, Hastings (Jan. 20).

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

GENERAL

Enucleation of the Bovine Corpus Luteum

Sir,—I can assure Mr. C. M. Gould that I read his letter (*Vet. Rec.* **71**. 855) very carefully, and nowhere in my response (*Vet. Rec.* **71**. 919) did I imply that "the condition is *necessarily* associated with cystic ovarian disease." In my letter I pointed out that in certain forms of cystic ovarian disease the subject, and hence its ovary, could be described as "anoestrous."

Yours faithfully,
F. L. M. DAWSON,
A.V.I.O. (Sterility).

Ministry of Agriculture, Fisheries and Food,
Veterinary Investigation Centre,
Madingley Road,
Cambridge.
January 19th, 1960.

Calcium Lactate as a Dietary Supplement for Laying Hens

Sir,—Some of your readers may remember the note published in THE VETERINARY RECORD (February 28th, 1959, p. 179) in which Worden and Reid claimed that by substituting calcium lactate pentahydrate for calcium carbonate (at 1 or 2 per cent. levels) they obtained "a significant increase in egg production, accompanied by a significant decrease in food consumption." In support of this work various statistical data were given, and "as a result of this promising result, further and more extensive trials" were planned.

Since the formula of the basal ration was given it was an easy matter to repeat these feeding trials which in the case of the Huntingdon workers related to 3 groups of 12 pullets. However formidable the statistician employed in such an experiment, I personally would not be prepared to place much reliance on a feeding trial using such a small number of birds in each group. However, that is by the way.

The claim was so outstanding, one felt the immediate need to repeat this work in the hope that comparable results would be obtained.

Trial I began on April 15th, 1959, with 47 13-month-old White Leghorn hybrid pullets, 26 receiving the basal mash and 21 the same ration plus 1 per cent. calcium lactate pentahydrate.* At the beginning of the experiment the experimental group were laying at a rate 4.31 per cent. higher than the controls and eating 5.82 per cent. less feed; but by the end (12 weeks later) there was a difference of only 3.24 per cent. in egg production and only 1.37 per cent. less feed was consumed.

It was felt that this failure to confirm the results of Worden and Reid might have been due to the age and type of pullets, so a second feeding trial was begun on July 29th, 1959, using 145 Light Sussex × Rhode Island Red pullets aged 8½ months. These were also housed in hen battery cages at Stoke Mandeville. The results were equally disappointing, for after 12 weeks the "lactate" group of 73 pullets were laying 10 per cent. fewer eggs and eating 2½ per cent. more feed.

One wonders why such a poorly designed hen battery formula was used for such an experiment. With 18 to 19 per cent. protein, and a theoretical energy content of 686 Cal. per lb. (Frapps Prod. Energy), the calorie/protein ratio is only 37.8:1, which is quite unsuitable for pullets laying at a level of 60 per cent. Furthermore, the cost of this basal ration at the time worked out at £34 18s. 9d. per ton, or about 3 guineas more than that for a good commercial hen battery ration. Incidentally, calcium carbonate costs only about £3 10s. per ton, whereas calcium lactate pentahydrate (in small quantities) costs 1s. 3d. per lb. or £140 per ton.

In these circumstances, and in terms of our results, we can see no place for calcium lactate pentahydrate as a substitute for calcium carbonate in layers' rations.

Yours faithfully,
W. P. BLOUNT.

2, Kingscote Street,
London, E.C.4.
January 18th, 1960.

* Bow-Caloe Ltd.

A Veterinary Television Talk

Sir,—The gentlemen who have responded favourably to my letter on the above subject seem to have a similar concern for the well-being of our profession. They have modified and added to my argument, and made suggestions that are quite reasonable and worthy of gentle discussion.

Not so your other correspondents. They have little concern for my suggestions. Yet a vital principle is at stake, a principle dear to the hearts of many. Mr. Roger Massey appears confounded that I should express an opinion, while Mr. F. W. Logue boggles at the possibility of my having one. In my ignorance, I overlooked the fact that a comparative practical inferiority is tantamount to a comparative intellectual inferiority. What I said cannot be of any significance.

In my former state, I would have said that these gentlemen, in their distasteful attacks, had lost track of what I wrote. Indeed, I would have tended to dismiss their letters. However, in my enlightenment, I must bow my head, and as Mr. H. Bell points out, must revert to my proper place and function. I do this, not with reluctance and rebellion in my heart, but happily, in the sublime knowledge that in 1984

I will be privileged to put forward profound opinions. Any resultant criticism I will counteract with the masterful thrust of, "I qualified in the fifties."

Yours faithfully,
D. H. PHILLIPS.

2, Horsley Hall,
Eccleshall,
Stafford.
January 17th, 1960.

Sir,—I wonder how many practising veterinary surgeons can claim that they have not been embarrassed as a result of the views expressed through this medium?

I personally have been confronted by parturition cases made unnecessarily difficult by delay in calling for assistance. I have often been compelled to agree with my clients that I do not subscribe to the views expressed. I am aware of more than one lost calf resulting from delay in giving assistance at parturition. Some of my clients speak very clearly and lucidly on this subject when they have allowed their better judgement to be overruled by "what they heard on television."

Yours faithfully,
J. P. PICKERING.

Orchard House,
Priestpopple,
Hexham,
Northumberland.
January 20th, 1960.

Animal Insurance

Sir,—Having a problem to which I can find no satisfactory solution, I wonder if I could air it through your columns?

I have a client who vaccinates all young animals against every possible thing (*e.g.* black-quarter, black disease, husk, abortion, anthrax, and so on). As these vaccinations cover the commonest causes of death in farm livestock, he has been trying without success to obtain an insurance either at a reduced rate on the production of a veterinary certificate saying that this programme has been carried out, or alternatively an insurance covering the risks of lightning and poisoning only. However, our insurance companies appear all to have their heads buried in the sands of arbitrarily fixed rates.

I am wondering what has happened to that doctrine of the customer being right, and if no business is really better than to make a change which could create a wide demand.

Yours faithfully,
W. J. TARVER.

39, Beech Grove,
Ashton-on-Ribble,
Preston,
Lancs.
January 20th, 1960.

Eclampsia in the Bitch

Sir,—I was interested to read Mr. Ascott's recent letter in THE VETERINARY RECORD under the title "Hypomagnesaemia in Small Bitches." He refers to the condition associated with hypocalcaemia in the bitch which is commonly known in this country as eclampsia and in North America as puerperal tetany.

So far I have been unable to trace Brooksbank's description of hypomagnesaemia in small bitches, to which Mr. Ascott refers, although I did find an interesting account by the same author (Brooksbank, 1932) entitled "Milk Sickness in Small Bitches." In this Brooksbank described the condition we know as eclampsia and suggested that it was analogous to milk fever in the cow. He also suggested that the blood chemistry would elucidate the problem. Incidentally, treatment at that time was by the subcutaneous injection of oxygen.

In an admittedly rapid search of the literature I have been unable to find any reference to hypomagnesaemia in the bitch, or to the blood magnesium levels in eclampsia. I should be most interested to learn of any work on blood magnesium levels in the eclamptic bitch.

One can explain the response to magnesium therapy in hypocalcaemia in the bitch; the action of the magnesium ion being to depress the central nervous system and thus to relieve the convulsions. On the same principle other and safer sedatives to the central nervous system have been used in treatment, *e.g.* the barbiturates and morphine.

Replacement therapy using calcium borogluconate solution by intravenous, intraperitoneal or subcutaneous injection is obviously preferable. Dihydrotachysterol can be used in the treatment of eclampsia, but I have no experience of its use in the bitch.

I would emphasise Mr. Ascott's last paragraph. Magnesium sulphate solution administered intravenously depresses the central nervous system. In fact saturated solutions of magnesium sulphate may be used by this route for euthanasia. Any intravenous injection of magnesium sulphate solution for therapeutic purposes must be made very slowly and the solution should be very dilute.

Yours faithfully,
JOHN STRATTON.

Department of Medicine,
Royal Veterinary College,
London, N.W.1.
January 21st, 1960.

References

BROOKSBANK, N. H. (1932). *Vet. Rec.* **44**. 417.

Persistent Right Aortic Arch in the Cat

Sir,—We have read with interest the report by Jessop of a Persistent Right Aortic Arch in the Cat (Jessop, 1960) and are prompted by it to record a similar case seen in this School on November 6th, 1957.

A male kitten between 10 and 12 weeks of age was referred to the hospital exhibiting the classical signs of constriction of the oesophagus by a vascular "ring," *viz.*: retention of liquid, but regurgitation of all solid food a variable time after ingestion; marked

debility, and retarded bodily development (when seen the kitten weighed only 12 oz.); gross dilatation of the oesophagus anterior to the base of the heart.

Unfortunately the kitten died before operation to relieve the obstruction could be performed. *Post-mortem* examination confirmed that the obstruction had been due to constriction of the oesophagus by a vascular " ring " and that this ring had been formed as a result of persistence of the right instead of the left fourth aortic arch.

We would also like to record our opinion that the final sentence of Jessop's description of his case does perhaps underestimate the obstacles to successful surgical correction of this malformation. Our experience of successful operation for this condition is limited to one dog, but in this case post-operative persistence of the dilatation of the oesophagus, with continued regurgitation of food, presented a far more serious problem than did the actual division of the ligamentum arteriosum and consequent relief of the obstruction. At present we can offer no solution to this problem, beyond emphasising the importance of operating on such cases as early as possible, before the dilatation has become excessive. If this is done it seems probable that the chances of the oesophagus returning to its normal size are likely to be substantially improved.

Yours faithfully,

S. W. DOUGLAS.
R. G. WALKER.
M. C. G. LITTLEWORT.
Department of Veterinary Clinical Studies,
School of Veterinary Medicine,
Madingley Road,
Cambridge.

January 22nd, 1960.

Reference

JESSOP, L. (1960). *Vet. Rec.* **72**. 46.

Displaced Abomasum in a Friesian Cow

Sir,—One of us was called to a recently calved Friesian cow on the evening of December 20th. A tentative diagnosis of displaced abomasum was made. It is of note that the animal had appeared quite normal that morning. The following morning the animal was re-examined by both of us and we decided to operate the next day, after 24 hours' starvation of the animal. On the 22nd the animal's condition had obviously deteriorated but we decided to carry out the operation using the method recently suggested by Mr. E. C. Straiton, with the exception that a solution of chloral hydrate and magnesium sulphate was used as anaesthetic. On opening into the abdominal cavity it was at once apparent that rupture of the abomasum had taken place with a massive peritonitis. The animal was therefore destroyed.

On *post-mortem* examination it was found that the abomasum had not ruptured from pressure as we expected but had in fact torn longitudinally through its greater curvature due to the extreme tension placed on the organ following its gut displacement.

We wonder if this has been the experience of other veterinary surgeons as it would appear that, in certain cases at least, one cannot allow the time delay for starvation prior to operating.

Yours faithfully,

K. A. PEMBREY,
L. R. STIRLING.
St. Mary's Close,
Church Green,
Witney,
Oxford.

January 4th, 1960.

" Progress "

Sir,—In their letters respectively of Jan. 2nd, and of Jan. 23rd, Messrs. Swift, and Robertson and Macleod are concerned at the agricultural client's present-day tendency to " do-it-yourself," helped and encouraged by a few producing and retail chemists.

While one accepts that a commercial company can only remain in business by making profits, one wishes that firms who sell direct to livestock owners could be brought to see that there is a matter of vital principle involved. In any case, they would be none the worse off by dealing exclusively with the profession while stock owners would be far better off if their animals were treated with medicaments prescribed after proper diagnosis. It seems to me with certain honourable exceptions, that those firms who have stood by us and " played the game " throughout the years consist mostly of the older-established firms who served us when our fathers were practising. We must, therefore, humbly thank those firms who have honoured the principle of selling their products solely to our profession.

For those who have, in our profession's opinion, turned against us, and forsaken us for the wide open plains of commercialism, the temptation has been understandably great, especially as we could not advertise their products for them: at least, not in the Press.

My opinion, to some extent, is that we ourselves have encouraged sales direct to the farmer by airily announcing that we are above " selling things as does a mere shop." Those of us who were born to this profession will remember the days our parents spent in making up hundreds of bottles of drugs prior to " epidemic seasons." One cannot help thinking that commonsense was an ingredient missed out when the cake was baked to raise the status of our profession in the last few years! How true is the proverb " pride cometh before a fall."

Well! We have had our pride, and here comes the fall in the form of large animal clients who come to us only when a beast is *in extremis*, with little hope remaining of curing it. If we do cure it the client will have two bills to pay, one from the veterinary chemist, and another from ourselves.

We therefore increase our examination fees (except in Cornwall!) to help pay for the lost discount on drugs, and for the raised salaries of our assistants, hoping this will remedy things. Not a bit of it: our farmer clients tend to cling, naturally enough, all the more to " do-it-yourself " methods.

What is the true, practical answer in order to put private practice back on a progressive basis?

I should say, for one thing, that we should press for an amendment of the present relevant Acts and Orders, so that no veterinary instrument or injectable drug may be purchased except from a veterinary surgeon; then we ourselves should be controlled by the R.C.V.S. as to what drugs or instruments may be supplied to clients for them to use on their own animals. A panel of veterinary surgeons in active practice should be formed to decide this, and also to decide exactly what minor surgical or other procedures a farmer may carry out on his stock. Things are getting slack in this respect. Every other profession or trade has full control of its own activities, and I feel we should make a decided attempt to pick up the reins of ours once more.

I would also like to see our newspaper made exclusive to our own profession; it is ridiculous to think that one cannot write a letter, intimate to our own affairs, without just anyone reading it.

As a temporary measure, I think we should not countenance any of our members taking up posts as advisers to firms who try to "short circuit" us by selling direct to retail chemists or farmers, and we should press very hard to make all firms quote us discounts for drugs.

In the small animal field much of our work is being done by inexperienced "semi-lay" people, and in most cases no attempt to conduct a system of almoning is made, with consequent heavy losses to our profession.

The answer here is that the poor client his sick animal, the various charitable institutes involved, and we ourselves could all be happy if an agreement over fees were made with these institutions, and an over-all voucher system evolved, to be worked only by local veterinary surgeons.

In the case of static clinics local veterinary surgeons could attend on a rota system charging so much per hour, but no resident surgeon should be allowed to be employed to the detriment of his local private colleagues.

In the main we have only ourselves to blame for letting things slip in the past: let us now prepare to stand up and fight for our profession in the future, and refuse to be pushed aside, down-trodden and ridiculed.

Yours faithfully,
R. HODDINOTT.

Mill Pond House,
Hayle,
Cornwall.
January 24th, 1960.

Sir,—The letters of Messrs. Swift, Robertson and Macleod raise important issues.

We might combat the the sales of medicines by "salesmen" by offering all necessary drugs at known retail prices and demand proper discount from our suppliers but how shall we retain contact with the farmers to prevent the unnecessary losses described by your contributors?

The Attested Herd Scheme has been very valuable in this respect: it enabled us to visit farms and to discuss farm problems in a friendly way.

In mid-Wales I think it helped to increase our work amongst sheep and our knowledge of sheep diseases.

The Montgomery Education Authority had an excellent scheme of adult education. "Fireside chats" were arranged for small groups of farmers where (for a small fee) veterinary surgeons and other agricultural experts would give talks. In this way farmers learned that the profession was interested in all the problems of animal disease and their prevention.

Does this suggest a line of approach?

Yours faithfully,
S. H. ADAMS.

125, Widmore Road,
Bromley,
Kent.
January 25th, 1960.

Griseofulvin in the Treatment of *Microsporum canis* Infection in Dogs

Sir,—I have read with interest the letter from Messrs. Ablett and Gill, in this week's issue of THE VETERINARY RECORD, relating to their experience with griseofulvin in the treatment of ringworm in a litter of puppies.

I have used griseofulvin* for the past 12 months in the treatment of ringworm in both cats and dogs. The first case on which we used the material was a Yorkshire terrier puppy of about 6 months old, and weighing approximately 3½ to 4 lb. The oral dose given was 250 mg. daily. This case had proved resistant to treatment with some of the usual topical ringworm dressings for a period of approximately 3½ months. After 3 weeks' treatment with griseofulvin little response was noted, and we contacted the manufacturers for advice. We were advised carefully to clip the hair over the lesions, which incidentally were chiefly on the head and neck, although there were one or two others on the limbs.

After further treatment for 21 days on the same dosage, together with the careful clipping of the hair referred to, this bitch was completely cleared, and has remained so ever since, a period of about 6 months. She has since had a litter of puppies, none of which showed any sign of ringworm lesions.

We have also treated a number of cats with griseofulvin, and two examples follow.

The first was a cat which had a ringworm lesion over the right eye. It proved resistant to topical applications for some weeks, but responded in approximately 6 weeks to an oral dosage of griseofulvin of 250 mg. daily.

The second cat was one which had had no other treatment; it responded to an approximately 6 weeks' course of the tablets.

All these cases were diagnosed as *Microsporum canis* by cultural methods.

Yours faithfully,
J. McCORMICK.

113, Oxford Road,
Reading.
January 23rd, 1960.

* Grisovin (Glaxo).

" Retained " Placenta

Sir,—Your correspondent's clinical note recalls to mind a somewhat similar experience concerning the longevity of the placenta in the bovine stomach.

The animal concerned was a 5-year-old Shorthorn cow which died suddenly 21 days after calving. *Post-mortem* examination revealed only a degree of denudation of the ruminal mucous membrane together with the presence in the rumen of the twisted and lumpy remnants, 3 feet long, of the placenta. The owner informed me that the cow had been in isolation since calving and that no sign had been seen of the afterbirth which had presumably been voided and ingested immediately after parturition, and had been churning around in the rumen for a full 3 weeks. On another occasion a similar disintegrating afterbirth was removed *via* rumenotomy in a 4-year-old Friesian cow 12 days *post partum* with beneficial results.

When one observes the delectation manifested by the post-parturient herbivorous cow whilst polishing off a large meaty placental mass all in one piece, it seems amazing that digestive upsets from this cause are not more frequent, and yet such happenings must be comparatively rare. I, too, would be interested to know from physiologists, how long it takes for the foetal membranes to be completely digested and whether other practising veterinary surgeons have encountered such complications at all frequently. If so, it would seem that, wherever possible, the removal of the offending mass out of sight of the cow at the earliest opportunity would be the most sensible policy.

Yours faithfully,
D. N. SPRIGGS.

The Grange,
Drove Road,
Swindon,
Wilts.
January 23rd, 1960.

SCIENTIFIC

A Short Trial Using Trimeprazine Tartrate as a Sedative and Premedicant in the Horse

Sir,—Over the last 6 months, 17 cases have been treated with 6549 R.P. tartrate to try to assess its value as a sedative and/or premedicant in the horse.

Method. In this series it was always given by the intramuscular route about 45 minutes prior to any minor treatment such as cauterising of warts, or before administering the general anaesthetic. The latter was usually chloral hydrate or thiopentone sodium often combined with local anaesthetic. Procedures ranged from neurectomies, roaring operations and firing to simple excision of warts.

Results. In order to simplify the results, I have divided the cases into 3 groups, namely:—

Group I. Nos. 5150, 4772, 5037, 4679, where the animals by temperament or training are docile to handling, hobbling, etc., and no conclusion was drawn regarding the drug's sedative effects. It appeared to have no adverse effect.

Group II. Nos. 4806, 5172, 4935, 4819, where it had no effect and even appeared to have an adverse effect in that the animal was more alert. No. 4806 is vicious if handled with a view to surgical interference or clipping, No. 5172 resented handling, No. 4935 reared on approaching or handling him, and No. 4819 was more nervous than normal.

Group III. No. 4902, 4684, 5034, 5091, 4840, 5061, 6700, 5075, and 5200 were cases where some benefit was seen from the use of this compound.

Taking all 3 groups into consideration, the following conclusions were drawn:—

1. A sedative effect was seen in only 9 of 17 cases and it was not marked. It appeared to be more "laziness" than a lack of awareness of surroundings.

In 8 cases there was no satisfactory effect and in 4 of these adverse effects appeared to be present as described in Group II.

2. The dose of chloral hydrate or thiopentone sodium was lessened in those cases where a general anaesthetic was used twice on the same animal, that is on one occasion without using the sedative and at a later date while using it.

3. Recovery time was not prolonged and the recovery was always tranquil even when using thiopentone.

4. Local tolerance was good: in only one case was the reaction other than very mild.

Discussion. Dosage in most cases was 5 c.c. of 5 per cent. solution which is slightly below the recommended dose of 0.4 to 1 mg. per kg. bodyweight for animals of 500 to 550 kg. bodyweight. In one or two cases, the dose was doubled or even trebled, and in the latter case we found a difference.

In case No. 5075, the animal had been anaesthetised on a previous occasion and was very violent, requiring 2½ oz. of chloral hydrate to anaesthetise it, to a fairly light degree.

Using 15 c.c. 5 per cent. solution of 6549 R.P. tartrate intramuscularly the animal submitted quietly to handling and required only slightly more than 1 oz. to anaesthetise it.

In the doses used, the sedative effect was not marked. Premedicating properties were satisfactory in that it reduced effective dose of anaesthetic and did not lengthen recovery time which was always tranquil even when using thiopentone sodium.

It is hoped to continue the trials using considerably higher doses intravenously and investigating the sedative effect more closely, as distinct from the drug's premedicating properties.

Acknowledgments.—I wish to thank Dr. L. W. Hall, M.A., who instigated the trial, for his helpful advice, and also May & Baker Ltd., for generous quantities of the drug.

Yours faithfully,
J. W. MACKAY,
Captain, R.A.V.C.

R.A.V.C. Training Centre,
Melton Mowbray,
Leics.
January 24th, 1960.